

The Signal Dynamics of Sensory Itch Signals in the Human Nervous System

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Introduction

Building upon previous work exploring the nature of pain signals and the important 2022 insight (ibid.) that pain is not the result of electrical signals emitted by nerves but the reflection of signals emitted by the brain which are reflected by molecules released from damaged nerves, this publication concerns the signal dynamics associated with itch.

Abstract

When a region of nerve cells becomes mildly irritated by a chemical irritant triggering an autoimmune response, itch is often the result. It is not the chemical, itself, which produces itch, however, but rather, the electrical reactivity of immune cells dispatched in response to the chemical irritation which produces this effect, I would posit. Itch can, importantly, occur even without chemical irritation. As circumscribed earlier this year, itch can be the result of non-touch sensory inputs, especially auditory inputs. The human brain seems to step up its sensitivity to the itch sensation in response to certain sounds, such as the sound of an insect's wings buzzing. This has obvious evolutionary origins. If a human ancestor was able to sense that a mosquito has landed upon its skin thanks to the aid of transiently hypersensitive touch sensation, that ancestor might more likely to survive as it could swat a mosquito before being bitten and perhaps contracting a disease such as malaria.

Skin can become itchy due to dryness, which provides further support for the autoimmune hypothesis. When living human tissues become desiccated, the ability of chemical signals to be properly expressed and for waste byproducts to be eliminated is impaired.

Based upon the fundamental assumption that pain is the result of chemical leakage from damaged nerves, it seems likely that chemical irritants resulting in itch must bring about this effect through a combination of both the leakage of small quantities of those chemicals associated with the production of pain signals along with the production of a signal reverberation which alters the pattern of the reflected signal received by the brain, resulting in altered perception.

Just as an altered pain signal emanating from the intestine can result in mood changes (e.g. in colic, PMS and tannen-containing alcohol-induced dyspepsia) as circumscribed in 31 March 2023, a different type of altered pain signal can result in the brain's unique perception of itch. The itch signal, being very similar to a faint pain signal, is the result of a combination of the low amplitude of the return signal and the conductive slowing of the signal by the very immune cells which caused the irritation in the first place. As the brain's mechanism for assessing the physical source of a pain signal is predicated

upon analysis of response time to what would, in computer parlance, be called a “ping,” any distortion to the feedback pulse in terms of timing or duration would result in uncertainty concerning the location of a signal source. When an itch is not scratched, the brain ceases to notice it after a few minutes, suggesting that the brain can eventually identify these confusing signals as noise and begin to filter the abnormal feedback. Scratching an itch temporarily relieves the sensation via the circulation of interstitial fluids, reducing the concentration of both Pain Signal Reflector Molecules and immune cells.

Whereas “referred pain” is the phenomenon by which pain is perceived as coming from a wholly different locus than its actual source, itch is, by contrast, the result of the rapid alternation between accurate and inaccurate perceptions of mild pain perception wherein there is a degree of signal referral but wherein the degree of this referral is minute. Itch is the byproduct of pain assigned by the brain to an incorrect source location by an offset of perhaps a centimeter or so. In traditional referred pain, trauma to regions of the body which traditionally are not injured create confusion due to identical ping times to other regions which happen to share the same distance from the brain and thus, the same ping time.

Conclusion

Steroidal ointments are often effective at relieving itch for reason that the itch sensation is the result of an autoimmune response to an irritation. Immune cells, as they send and receive information ionically, are likely culprits in the reverberation effect resulting in the perception of itch. This effect could be likened to a musician holding a note for just slightly too long a length of time, creating confusion in the audience as to whether it as an intended improvisation or simply an error. An improved physical and logical understanding of something as banal as itch could provide new insights into the study of diseases of the nervous system and a new area of focus for future studies.